



Case report

Subarachnoid hemorrhage caused by a traffic accident: De novo aneurysm ruptured 30 years after surgical neck clipping



Atsushi Yamada, M.D., Graduate Student, Kana Unuma, M.D. Jr Assistant Prof.,
Haruka Kojima, BPharm, Graduate Student, Koichi Uemura, M.D. Prof.*

Department of Forensic Medicine, Graduate School of Medical and Dental Sciences, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113-8519, Japan

ARTICLE INFO

Article history:

Received 14 August 2013

Received in revised form

3 November 2013

Accepted 7 December 2013

Available online 21 December 2013

Keywords:

Subarachnoid hemorrhage (SAH)

Traffic accident

Surgical neck clipping

De novo aneurysm

Wall shear stress (WSS)

ABSTRACT

A man, who had a medical history of surgical neck clipping 30 years previously, died of traffic accident. The medico-legal autopsy showed subarachnoid hemorrhage and ruptured aneurysm on the lateral side of the clip. Microscopic examination showed the aneurysm was not to be regeneration, but a new de novo aneurysm. We diagnosed the cause of death was traumatic aneurysmal rupture. In addition, we discussed the cause of a newly formed de novo aneurysm which may be affected by past surgical neck clipping.

© 2013 Elsevier Ltd and Faculty of Forensic and Legal Medicine. All rights reserved.

1. Introduction

Subarachnoid hemorrhage (SAH) in patients with known surgical treated pre-existing aneurysms is a recognized but rare phenomenon,¹ and rarely seen at post-mortem. Generally, surgical neck clipping is regarded as a radical cure of aneurysmal rupture. Tsutsumi et al.² reported that aneurysmal regrowth was detected in 2.9% of clipped aneurysms on long-term observation. Residual neck aneurysms are the main causes of regrowth. Residual neck aneurysms result from inaccurate placement of the clip, or partially or completely off.^{3,4} On the other hand, if neck clipping is performed completely, the annual rate of regrowth is 0.26–0.52%. De novo aneurysms account for 0.9–1.1% of all ruptured aneurysms which are successfully treated.⁵ In practice, the etiology of the de novo aneurysm is not fully understood.

We describe here a case of traumatic rupture of cerebral saccular aneurysm on the lateral side of the clip placed 30 years previously. In addition, we discuss the cause of a newly formed de novo aneurysm which may be affected by the past surgical neck clipping.

2. Case report

2.1. Case history

A 74-year-old man was struck by a passenger car (20 km/h) and strongly hit his forehead on the road. Soon, he fell into unconsciousness and cardiopulmonary arrest. Immediately transferred to an emergency hospital. An urgent computed tomography (CT) scan showed severe SAH, and there was a metal artifact around the left chiasmatic cistern (Fig. 1A). Three-dimensional CT angiography (3D-CTA) revealed an intracranial aneurysm and a clip (Fig. 1B(a,b)). Although spontaneous cardiac beating appeared, he died 16 h after admission. The clinical diagnosis was traumatic SAH. Judicial autopsy was performed 46 h after his death.

2.2. Autopsy findings

The deceased was 165 cm tall and his body weight was 58.7 kg (body mass index: 21.6 kg/m²). Skin abrasions were present in the right orbital margin to the forehead with small blood clots. No other noteworthy injuries were found. No signs of fracture of the cranium existed. At the bottom of the brain, almost symmetrical SAH was extensively distributed around the left temporal lobe. At the origin of the left anterior cerebral artery, a saccular aneurysm that measured 0.8 cm in diameter with a bulging hematoma existed,

* Corresponding author. Tel.: +81 3 5803 5199; fax: +81 3 5803 0128.

E-mail address: kuemura.legm@tmd.ac.jp (K. Uemura).

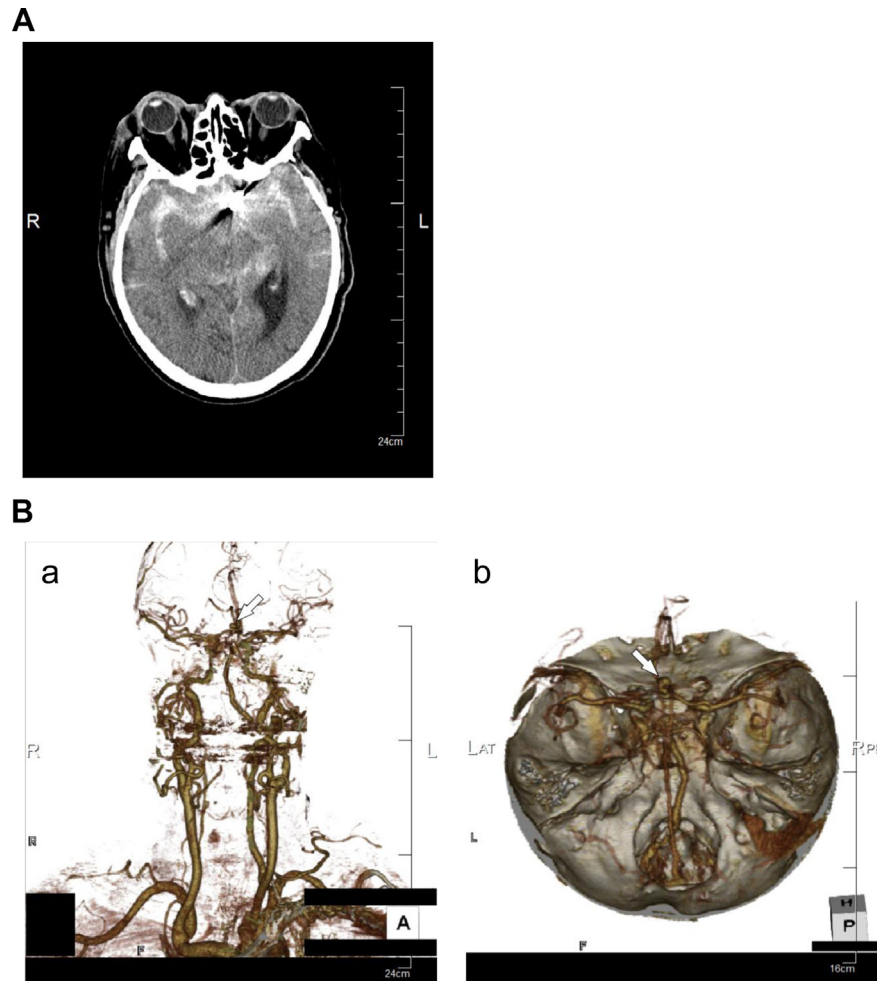


Fig. 1. Brain CT images. **Panel A.** An axial CT image showing subarachnoid hemorrhage with a metal artifact around the left chiasmatic cistern. **Panel B.** (a) A frontal view of a 3D-CTA image revealed a left ACA intracranial aneurysm lateral to the clip. The white arrowhead shows the aneurysm. (b) A top view of the 3D-CTA image. The white arrowhead shows the aneurysm.

which was thought to be a ruptured aneurysm. The clip was 1.0 mm proximal to the aneurysm (Fig. 2A).

2.3. Histopathological findings

Around the ruptured aneurysm, the parent artery was investigated histopathologically in a sequence (Fig. 2B). Just before the aneurysm, beneath the clip, intimal hyperplasia existed with thinning of the internal elastic laminae and tunica media (Fig. 3A). The ruptured saccular aneurysm wall was composed of only fibrous tissue and fragments of the muscular coat. In the aneurysm, partially clotted blood and no fibrotic changes were seen (Fig. 3B). At the vertebra-basilar arteries, there was mild atherosclerosis.

3. Discussion

In our case, the ruptured aneurysm was on the lateral side of the clip placed 30 years previously. So, we needed to investigate whether the aneurysm was regrowth or regeneration. At the clipped site, the previous aneurysm was completely organized. Therefore, we regarded it as a newly formed, de novo aneurysm and the aneurysm ruptured due to head trauma. The potential causes of de novo aneurysm are considered as follows. Past surgical manipulations might cause vascular wall injuries. Congenital medial

defects and atherosclerosis weaken the vessel wall. One of the multiple aneurysms might have been overlooked. Risk factors such as smoking, hypertension and a history of migraine contribute to increase aneurysm growth and even more rupture.⁶ Particularly, hemodynamics are believed to play the most important role.

Recently, the wall shear stress (WSS) forces have been implicated as a regulator of vascular wall remodeling. Under a high WSS, endothelial cells exhibit high cell division rates and a low cell density. Alternatively, a low WSS results in a thinning of the medial layer because of a smooth muscle apoptotic mechanism.^{7,8} Proper tensile states are essential to maintain normal vessel wall behavior. Sumi et al.⁹ demonstrated that vascular narrowing weakened the WSS in the flow reattachment region.

In our case, the ruptured aneurysm occurred 1.0 mm distal to the clip. Beneath the clip, vascular narrowing was noted histopathologically. Vascular narrowing was caused by intimal thickening. Mechanical stimulation promotes vascular smooth muscle cell proliferation, migration through the internal elastic lamina, and the deposition of extracellular matrix proteins. A clip could be a stimulus for the following reasons. Surgical manipulation, inappropriate clip placement, oppression of the clip edge, and clip rotation may lead to distortion of the arterial wall.¹⁰

In conclusion, we showed a case of de novo aneurysm generation 30 years after surgical neck clipping and discussed the possible

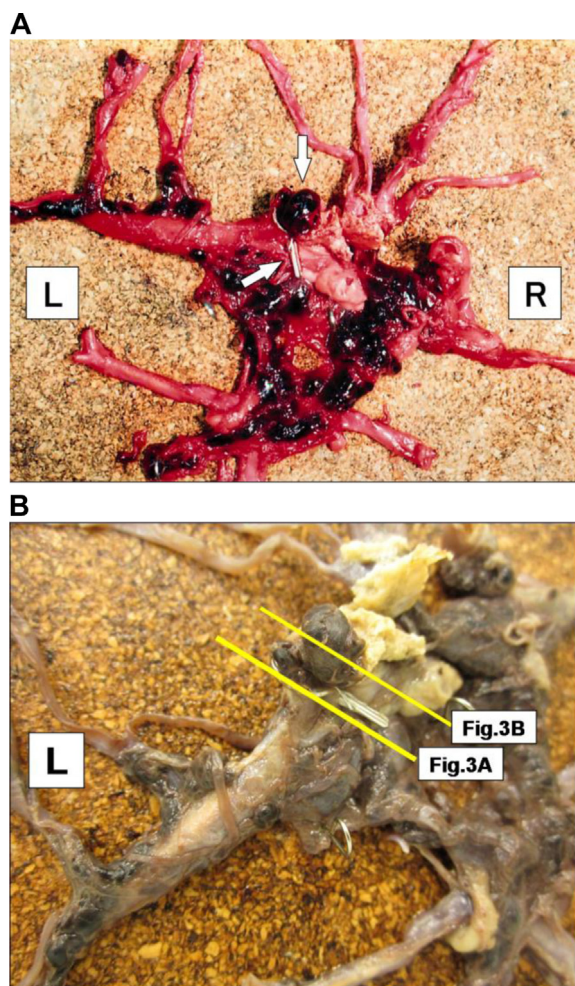


Fig. 2. Macroscopic observation of the aneurysm. **Panel A.** Cerebral arteries. Origin of the left ACA, a ruptured aneurysm with bulging hematoma. The clip was located about 1 mm proximal to the aneurysm. **Panel B.** Formalin-fixed cerebral arteries. Histo-pathological investigation of the parent vessel in a sequence.

contribution of past surgical neck clipping which caused flow dynamic change and vessel wall deformation. Careful observation of the vascular morphology around the aneurysm may be informative for forensic pathologists.

Ethical approval

None declared.

Funding

None declared.

Conflict of interest

All the authors declare no conflict of interest.

References

1. Rothmeyer S, Lefevre D, Taylor A. Recurrent or new symptomatic cerebral aneurysm after previous treatment. *Interv Neuroradiol* 2005;11(4):341–8.

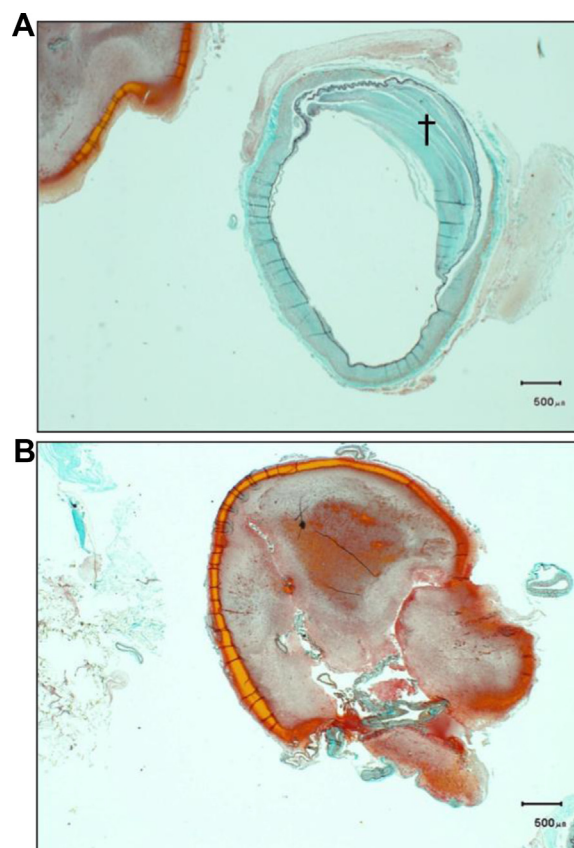


Fig. 3. The histology of the aneurysm. **Panel A.** Just before the aneurysm, beneath the clip, (†) intimal hyperplasia existed with thinning of the internal elastic laminae and tunica media (Elastica-Masson Goldner stain $\times 20$). **Panel B.** The ruptured aneurysm (Elastica-Masson Goldner stain $\times 20$).

2. Tsutsumi K, Ueki K, Morita A, Usui M, Kirino T. Risk of aneurysm recurrence in patients with clipped cerebral aneurysms: results of long-term follow-up angiography. *Stroke* 2001;32(5):1191–4.
3. Drake CG, Friedman AH, Peerless SJ. Failed aneurysm surgery. Reoperation in 115 cases. *J Neurosurg* 1984;61:848–56.
4. Juvela S, Poussa K, Porras M. Factors affecting formation and growth of intracranial aneurysms: a long-term follow-up study. *Stroke* 2001;32(2):485–91.
5. Bruneau M, Rynkowski M, Smida-Rynkowska K. Long-term follow-up survey reveals a high yield, up to 30% of patients presenting newly detected aneurysms more than 10 years after ruptured intracranial aneurysms clipping. *Neurosurg Rev* 2011;34(4):485–96.
6. Vlak MH, Rinkel GJ, Greebe P, Algra A. Risk of rupture of an intracranial aneurysm based on patient characteristics: a case-control study. *Stroke* 2013;44(5):1256–9.
7. Dardik A, Chen L, Frattini J. Differential effects of orbital and laminar shear stress on endothelial cells. *J Vasc Surg* 2005;41(5):869–80.
8. Chatziprodromou I, Tricoli A, Poulikakos D. Haemodynamics and wall remodelling of a growing cerebral aneurysm: a computational model. *J Biomech* 2007;40(2):412–26.
9. Sumi T, Yamashita A, Matsuda S. Disturbed blood flow induces erosive injury to smooth muscle cell-rich neointima and promotes thrombus formation in rabbit femoral arteries. *J Thromb Haemost* 2010;8(6):1394–402.
10. Hayashi Y, Kimura M, Satake R, Kinoshita A. Possible participation of clip rotation in the formation of de novo aneurysm. *J Clin Neurosci* 2004;11(3):331–4.